REVIEWS.

Art. XX.—*De la Fièvre Bilieuse Melanurique des Pays Chauds comparée avec la Fièvre Jaune. Etude Clinique faite au Sénégal.* Par L. J. B. Berenger-Feraud, Médecin-en-Chef de la Marine, etc. etc. 8vo. pp. 434. Paris: Adrien Delahaye, 1874.

The Melanuric Bilious Fever of Warm Climates compared with Yellow Fever. A Clinical Study of the Disease as observed in Senegambia. By L. J. B. Berenger-Feraud, etc. etc.

Hemorrhagic Malarial Fever. An address delivered before the Medical Society of North Carolina, at its 21st Annual Meeting. By Wm. A. B. Norcom, M.D., President of the Society. Pamphlet. Raleigh, 1874.

Report on a New Variety of Malarial Fever. By T. C. Osborn, M.D. The New Orleans Journal of Medicine, Oct. 1868.

Essay on Malignant Congestive Fever. By J. D. Osborn, M.D. Ibid. January, 1869.

Hemorrhagic Malarial Fever. By R. F. Michel, M.D. Ibid. July, 1869.

Remarks on Malarial Hæmaturia. By S. F. Starley, M.D. Ibid. Oct. 1869.

Brief Account of an Epidemic of Jaundice. By Prof. L. S. Joynes. The Richmond and Louisville Medical Journal, April, 1868.

Miasmatic Hæmaturia. By William A. Greene, M.D. Ibid. Feb. 1872.

Hæmaturia or Yellow Remittent. By Edward H. Sholl, M.D. Medical and Surgical Reporter, Oct. 24, 1868.

Hemorrhagic Malarial Fever in Alabama. By E. D. M'Daniel, M.D., of Camden, Ala. Reprinted from Transactions of the Medical Association of the State of Alabama, 1874, 8vo. pp. 27.

Many of the Southern medical journals have during the last ten years contained descriptions of a disease which was unknown to most of those under whose observation it came. Its prominent and characteristic symptoms are jaundice, great irritability of the stomach, giving rise to uncontrollable vomiting, and a peculiar alteration in the colour of the urine, generally attributed by writers on the disease to the presence of blood. These have also with very few exceptions correctly regarded it as a manifestation of malarial poisoning; by some, however, it has been thought to be a modification of yellow fever. The admirable work of M. Berenger-Feraud will enable us, we think, to show not merely that the disease is not new, but also that the foreign body to which the urine really owes its remarkable appearance is bile, neither blood-corpuscles nor hæmatin being found in more than a very small proportion of the cases.

M. Berenger-Feraud's position as Médecin-en-Chef de la Marine, at once entitles him to consideration at our hands. In addition to this he seems to have enjoyed exceptional advantages for the study of the disease, having been for many years in charge of a hospital at Gorée which received annually into its wards from eleven to thirteen hundred patients, of whom on an average from thirty-five to forty were affected with melanuric bilious fever. At a later period he was physician in chief of the colony of Senegambia and of its dependencies, a position which brought him in contact with every class in the community, and enabled him to see the disease under every variety of circumstances. He has in this way been able to collect notes of at least three hundred cases; a larger number probably than has passed under the observation of any other physician. Not only has he been careful to record all that throws light upon the pathology of the disease, but he has also spent much time and labour in rendering it clear that it has no connection of any kind with yellow fever. Debarred, as he was, by the remoteness of the colony in which he was stationed, from the use of libraries, his book will not be found to contain many references to other authors. It is based, as he says, upon the results of his own observations, and, regarded in this light, it is an exceedingly valuable contribution to the literature of the subject of which it treats.

Without further prelude, we shall at once proceed to consider M. Berenger-Feraud's definition of the disease. It is, he says, a malarial fever of variable type characterized by—1. Persistent vomiting of bilious matters of a greenish colour; 2. Jaundice of the skin and of all the tissues; 3. A peculiar brownish colour of the urine. The second and third of these characteristics are more pathognomonic than the first, and when both are present enable us readily to distinguish this form of malarial disease from every other.[1]

M. Berenger-Feraud proposes a new name, Melanuric Bilious Fever, for this disease. He does this because the names hitherto applied to it are objectionable, since they imply either that it is a modification of yellow fever, or that the peculiar discolouration of the urine is due to an intermixture of blood. The name he has chosen, while it recognizes the malarial origin of the disease and the change in the urine, does not necessarily commit him to any theory in regard to the cause of the latter.

The second chapter of the book is devoted to the history of the disease. We have already intimated that our author was not disposed to look upon the disease as a new one. As the discussion into which he enters occupies nearly sixty pages of his work, we can adduce a few only of the many reasons which he gives for his opinion, which is sustained by the experience of several American physicians. Thus Dr. T. C. Osborn says:—

> "Here and there, in the several locations I have taken, during the twenty-eight years of my professional career, it has occurred to me to see, now and then, after long intervals of time, isolated instances having the same general features and results, but, classing them together under the congestive form, I failed to recognize the important position, in the family of malarial fevers, to which they were unquestionably entitled."

Dr. A. G. Mabry[2] in a report on a case of this disease writes:—

> "It is a mistake to suppose that this is a new form of disease. More than twenty-five years ago I treated, in the vicinity of Selma, cases of intermitting

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[1] This disease must not be confounded with that described, among others, by Drs. Harley, Dickinson, Greenhow, Gull, and Pavy, and called by them intermittent or paroxysmal hæmaturia, or, more properly, hæmatinuria. The exciting cause of the latter seems to be exposure to cold, and it has been observed in individuals who have never visited malarious regions. Jaundice has occasionally occurred in hæmatinuria, but it is by no means so marked and constant a symptom as in melanuric bilious fever, and the same is true of irritability of the stomach, which seems scarcely to exist in the former disease.

[2] Transactions of the Medical Association of Alabama, 1870.

fever presenting, in a marked degree, all the symptoms characteristic of these cases at the present day."

A very similar opinion is expressed by Dr. E. D. M'Daniel, who quotes passages from the writings of Dr. James Copeland,[1] Dr. R. B. Todd,[2] M. Segaud,[3] Dr. J. C. Guardia,[4] Dr. J. C. Faget,[5] and others, all of which sustain the position he takes.

We may add here in passing that a careful examination of the cases reported by Morehead[6] has convinced us that two or three of them are really instances of this disease, although they are not so described by him. But to return to the arguments presented by M. Berenger-Feraud. He says, first, that the physical conditions of the country in Senegambia justify the belief that the disease has always existed, although undoubtedly it may have been more virulent at some times than at others. The writers who hold the opposite opinion assert that the action of the malarial poison is modified by the epidemic influence which produces yellow fever, and that, as a result of this modification, a hybrid disease is produced. In order to be able to decide this point, M. Berenger-Feraud consulted the records of the hospital at St. Louis, and, although these are not so full as might be wished, he thinks he has found sufficient evidence that the disease existed as early as 1820, long prior to the appearance of yellow fever in Senegambia. The records of the hospital at Gorée also afford corroborative evidence of the correctness of his view. Moreover, neither Dr. Thèze nor Dr. Loupy, both of whom practised in Senegambia about 1856, seem to have regarded it as new, since they described it among the endemic diseases of the country. The author's conclusions in reference to this point are as follows: 1. Melanuric bilious fever is endemic on the coast of Africa, and is a manifestation of malarial intoxication. 2. It not only does not arise from the same cause as yellow fever, but also is not the result of a modification by the epidemic influence causing the latter. If it has only been observed at certain times and in certain localities in Senegambia, this is simply because the European inhabitants are fortunately not at all times placed in conditions favourable for its development. It has, therefore, been observed to be especially frequent at times when expeditions were made into the interior of the country, and consequently into malarious districts. It is rarely seen in a European during the first year of his residence in the colony; it is more common in the second, and still more so during the third; but after this the liability to be attacked by it apparently diminishes. Thus of 100 cases, 5.4 will occur among residents of one year, 22.5 among those of two, 42.5 among those of three; while the tendency to it after the third year may be represented as 20 per cent. for the fourth year, and 4.8 per cent. for the fifth. This apparent immunity is explained by the fact that very few foreigners pass more than three years in Senegambia. Prolonged residence, therefore, does not diminish the liability to this disease; on the contrary, its victims are to be found among those who have suffered from repeated attacks of malarial disease, and in this point M. Berenger-Feraud's experience is confirmed by that of several physicians, resident in the Southern States of our own country, who have written on this disease and whose names head this notice. There is in this respect a marked difference between this disease and yellow fever, which when epidemic in a locality is especially prone to attack new-comers.

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[1] Dictionary of Practical Medicine.

[2] London Med. Gaz., Jan. 19, 1849.

[3] On the Climate and Maladies of Brazil, 1845.

[4] Medicine through the Centuries.

[5] New Orleans Journal of Medicine.

[6] Clinical Researches on Disease in India, London, 1856.

It is also true that, while the former is endemic in parts of the country which have never been invaded by the latter, this has presented no deviation from its usual course when occurring in malarious districts.

The next chapter treats of the pathological anatomy of the disease. The author thus describes the appearances presented at the post-mortem examination in a typical case. The skin is uniformly yellow, and does not present, as in yellow fever, a more intense discolouration in some regions than in others. The depth of the colour depends, to some extent, upon the period at which the patient has succumbed to the attack, being more marked at first. There may be ecchymoses, but these are not so common as in yellow fever, and the effusions of blood into the muscles, so frequent in the latter disease, are not met with in this. The mucous membranes are not swollen, and there is no escape of blood from the anus, mouth, or nose. Nothing characteristic is found in the cranial cavity, and nothing beyond mere hypostatic congestion of the lungs in the thoracic. The stomach is full of a greenish liquid, either clear or similar in appearance to water in which spinach has been boiled, and entirely different in appearance from the material composing the black vomit, and is due simply to a reflux of bile. The mucous membrane of the stomach is healthy, being, however, of course, stained by bile. Other writers have described various morbid appearances, which M. Berenger-Feraud is sure are more correctly attributed to the effects of alcoholism; drunkards furnishing a large proportion of cases of this disease. The appearances alluded to consist in injection of the mucous membrane, disseminated ecchymoses, projection of the follicles, and opacity of the epithelium, lesions which are found in other diseases. The intestines are either healthy, or present no marked peculiarity. The liver, on the other hand, is often the seat of decided changes, being usually notably enlarged. Estimating the weight of the liver in health at 1.796 grammes, which is above that usually given, he finds its weight in this disease increased by from 200 to 1000 grammes. This increase is largely due to congestion, and the liver will therefore weigh much less after free incisions have been made into it. Its colour depends to some extent upon the stage at which death has occurred, being dark in cases that ended fatally in the early stage, and light in those that have lasted longer. Hence has arisen, the author says, the circumstance that some writers describe the liver as engorged with blood, and others as anæmic. The congestion is more marked in some parts than in others, and, consequently, the colour is not uniform. This gives the liver a marbled appearance. The blood which escapes from its cut surface is black, with a peculiar violet shade, due to the intermixture of bile. The liver feels hard to the touch, but its consistence is diminished. The gall-bladder is distended with a dark-coloured viscid bile, which stains the fingers like iodine. It is scarcely necessary to add that these are not the post-mortem appearances we meet with in yellow fever, in which the liver is of a pale-yellow colour, anæmic and dryish, and in which the gall-bladder contains a serous colorless or greenish liquid.

The spleen is almost always increased in size, weighing usually twice, and sometimes three times as much as usual. In the early stages of the disease it is almost diffluent; later it is hard. The kidneys are congested and their weight is also increased. Any alteration in structure which may have existed was not positively ascertained in consequence of the want of the necessary instruments to make a complete examination. There is reason to believe, however, that sometimes permanent injury is done to the kidneys by the fever. The character of the urine found in the bladder varies with the stage of the disease. The blood contains bile in sufficiently large proportions to stain linen yellow when dipped into it.

The American physicians, to whose papers we have already referred, seem to have enjoyed very few opportunities for the study of the post-mortem appearances, but Dr. Michel's and Prof. Jones's examinations,[1] so far as they went, revealed the presence of almost identical lesions with those given above. They both speak of the congestion of the liver and of the distension of the gall-bladder with bile, upon which so much stress is laid by M. Berenger-Feraud. Prof. Joynes also alludes to the proofs found at the autopsy, that there has been a free secretion from the liver during life. In his own words, "bile is found *deluging the body.*" In the face of evidence such as this it can scarcely be longer maintained, as has been done by many physicians, that torpor of the liver is an important factor of this disease.

The author next passes to the consideration of the course the disease generally takes. We have already said that it selects its victims from among those who have repeatedly suffered from malarial fever. More than this, it very rarely occurs witout premonition, since the melanuric paroxysm is generally preceded by others, and it is exceedingly important to be able to recognize it during the forming stage, when it may sometimes by proper treatment be averted. Unfortunately this can rarely be done with certainty. The first fully-formed paroxysm is ushered in by a chill, which is both of longer duration and of greater severity than usual, and this occurring where the disease is endemic, should at once awaken suspicion. During the intermission that follows the patient suffers from malaise and from a saburral condition of the *primæ viæ.* The second paroxysm occurs twenty-four hours after the first, and this is more severe, and is generally, but not invariably, accompanied by characteristic symptoms such as nausea, vomiting, cephalalgia, and melanuria. Sometimes, as this paroxysm is passing off, jaundice will be observed. The type of the disease may be intermittent, remittent, or continued. It may vary in degree to such an extent that M. Berenger-Feraud thinks it worth while to make four varieties: 1, mild; 2, severe; 3, grave; 4, *siderante* or pernicious. The type of the mild form is generally intermittent, that of the severe form, remittent, and that of the graver forms is continued; but this rule is by no means absolute, since a grave case may be of the intermittent type, and a mild case of the remittent. Since the difference between these four varieties is entirely one of degree, it will not be necessary to describe the symptoms of each variety in detail. In addition to jaundice, vomiting, and melanuria, the patients will generally complain of a feeling of weight and pain in the right hypochondrium, extending to the epigastrium and to the regions of the kidneys. The same regions are tender on pressure. Sometimes this pain is so great that it constitutes one of the principal features of the disease. The jaundice is uniform, and is very rapid in its appearance. Dr. J. D. Osborn says of it, that the nurse will very frequently be able to tell to a minute the time of its occurrence. Attention is generally called to the condition of the urine by the patient himself. Its colour varies usually from a light to a dark chestnut; it may be almost black in bad cases. Directly after excretion it is clear, containing neither deposit, nor mucus held in suspension. Afterwards its lower strata become muddy and a little grayish in colour. In severe cases, on the other hand, the urine, even when freshly passed, is turbid. Micturition is easy. In the mild variety the quantity of urine is increased, as much as three quarts being sometimes passed during the first paroxysm.[1] In the severer forms, however, there is a diminution of the secretion, but this never amounts to true suppression. It foams readily upon agitation, is acid at time of emission, but rapidly becomes neutral, or even alkaline, when allowed to stand. It contains albumen in varying proportions, but its amount does not bear any fixed relation to the gravity of the attack. It occasionally also contains casts, and has usually a specific gravity of 1026. During the intermission the urine loses its colour, becoming like that secreted in anæmia. The tongue is large, pale, and covered with fur, which is thick and white, excepting when discoloured by the matters vomited. Thirst is often intense, but the patient fears to drink lest vomiting should be provoked by it. The matters first thrown up consist of articles of food, afterwards pure bile is vomited. This is at first yellowish-brown in colour, but later it becomes a porraceous green. It is clear in mild cases, imparting only a light-greenish stain to linen dipped into it, and contains little or no solid matter. In the severer forms, however, the ejecta contain particles of solid matter, green in colour. M. Berenger-Feraud insists strongly upon the fact that the colour of the vomited matters is always green, unless altered by something which has been taken into the stomach; having never seen anything which at all resembles black vomit, he does not believe that it ever occurs in this disease. Prof. Jos. Jones, on the contrary, asserts that it is occasionally observed, but unfortunately we can only quote him at second-hand, and do not know, therefore, whether or not his assertion rests upon microscopical examinations. In severe cases there is, in addition to the symptoms above detailed, distressing headache. This is never localized in the supra-orbital region, as in yellow fever, but is diffused and more contusive in character than the pain in that disease. In mild cases the bowels are not disturbed; in the severer forms diarrhœa of a bilious character may occur. At first the stools are fecal, especially if there has been constipation, but later they become liquid, and so closely resemble the urine that they cannot be distinguished from it, and have been thought, even by physicians, to contain blood. In very bad cases hiccough is added to the other symptoms. It is usually distressing to the patient, and when it occurs, especially if accompanied by other nervous symptoms, it indicates great prostration, and generally a fatal termination of the case. American writers have described petechiæ and sanguineous discoloration of the liquid of blisters as of frequent occurrence in this disease. These the author has never seen, although he admits they have been observed by other physicians.

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[1] Quoted by Norcom.

It will be noticed that in the foregoing description there is no allusion made to hemorrhage from the gums and nose, or from other mucous surfaces, and while all the writers in the American journals whom we have already quoted speak of this as happening very frequently, they report very few cases which sustain them in this statement. Thus, among the ten cases narrated by Dr. T. C. Osborn, it is recorded in only one. Since it is also mentioned that vibices were present, it is at least possible that the oozing of blood from the gums depended upon a depraved condition of the blood not wholly ascribable to this disease.

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[1] Dr. S. F. Starley, after expressing the opinion that the disease is a congestive form of remittent fever, the stress of which is thrown upon the kidneys, writes as follows: "Certain it is that they are excited to greatly increased functional activity, for, coincidently with the hemorrhage, the quantity of urine secreted is largely above what I have ever known in any other condition of the system, unless it is when the kidneys take upon themselves the task of removing some large dropsical effusion."

The duration of the disease varies greatly. In the pernicious form it may prove fatal in three days. In the other forms, when death occurs, it is longer delayed. Even in mild cases that do well it is about three weeks before convalescence is established, and in other cases it is much longer, being in bad cases as much as sixty-five days. The mode of termination of every case will depend, to a large extent, upon its severity, although the results of cases treated by quinia are of course more favourable than those treated without it.

In the mild form recovery almost invariably takes place; in the severe form the mortality may be estimated at 20 per cent.; in grave cases the chances of life and death are about even, in the remaining forms death is the rule.

Complications occurring in this disease may be divided into two classes: 1, those observed during the disease itself; and, 2, those observed during convalescence. Under the first head is to be included a paroxysm of the gravest forms, coming on suddenly and unexpectedly in the course of a mild attack. Convalescence is very apt to be interrupted by, 1, gastric derangements, such as indigestion, nausea, vomiting, etc.; or, 2, by those of the intestines, among which colic, constipation, and dysentery are the most frequent. In addition to these, hepatitis, erysipelas, and paralysis are mentioned as occasionally present. Amblyopia has been sometimes noticed, and is probably due simply to anæmia of the retina, or to some disorder of accommodation.

Relapses are frequent, and it is often impossible to assign any reasons for them, but it is certain that their liability to occur is in inverse proportion to the efficiency with which the quinia treatment has been carried out. Indeed the tendency to relapse is so great that M. Berenger-Feraud says that any one who has once suffered from the disease in a severe form had better leave the country, since a succeeding attack, which is almost certain to occur, will probably terminate fatally.

When speaking of the etiology of the disease, the author says statistics show that the largest number of cases occur in the most malarious regions or in districts having a luxuriant vegetation. High temperature favours vegetable decomposition, but further than this it seems to exert no influence. The disease is most frequent during those seasons when it is reasonable to suppose that malaria is most active. It is occasionally met with, it is true, during cold weather, but in these cases the patient is generally an old sufferer from malarial fevers, in whom there is a strong tendency to relapse which has been probably called into activity by exposure to an abrupt change of temperature, or perhaps by a change of residence. It is well known in this country that either of these causes will sometimes provoke a recurrence of intermittent fever. Entire freedom from all liability to the disease is possessed by no race. It is true that the native inhabitants of Senegambia rarely suffer in their own homes, but when removed from them they also succumb to its causes. This may, partly at least, explain the immunity from malarial disease which is claimed for the blacks in our Southern States; although it is only fair to add the existence of this immunity has been recently called into question. Among predisposing causes to the disease may be mentioned insufficient and unwholesome nourishment, which acts by enfeebling the individual and by diminishing his powers of resistance. The excessive use of alcohol is also a predisposing cause. Drunkards not only eat, as a general rule, insufficiently, but are sufferers in a larger proportion than others from affections of the liver, which, of course, gives malarial disease, when it exists, a tendency to this form rather than to any other. So frequent indeed is it to find the changes produced by alcohol at post-mortem examination of individuals who have died of this disease, that, as we have already said, alcoholic gastritis has been described as one of the lesions it produces. Sometimes alcohol appears to act as an exciting cause. "Nothing is more common in Senegambia than to see an individual who has been drunk for two days, presenting on the third the premonitory symptoms of the disease." In concluding his remarks upon etiology, the author says that melanuric bilious fever is the result of malarial intoxication, producing a marked polycholia, that the symptoms resemble those caused in animals by the injection of bile into the blood, and that malarial disease assumes this form partly at least because of the frequency of affections of the liver, or of a tendency to them which exists in hot climates.

If the disease be malarial in its origin, why has it been so infrequent in our Southern States until within the last ten years? This is a question which it is not easy to answer fully. Dr. Norcom attempts to do so as follows:—

> "Before the war, the Southern States were in a high state of cultivation, and the lands thoroughly drained; hence the malignant forms of malarial disease as a general rule were not known, except in very low, badly drained swamp lands. Within the past eight years, owing to so much land lying waste, defective drainage, and the general unsanitary condition of the country, the malarial poison has acted with intense virulence, and caused the disease we are now considering. Since the prevalence of this disease we scarcely ever hear of the old algid pernicious fever."

A very similar opinion is expressed by Dr. Greene, while Dr. M'Daniel contends that it is due solely to increased virulence of the epidemic influence.

The diagnosis of this disease generally involves no difficulty. It rests mainly upon the recognition of the peculiar alterations in the colour of the urine. We have already described its microscopic characters so fully that it is not necessary to do this again. We shall, therefore, proceed at once to give the results of the chemical and macroscopical examination. Nitric acid will, in addition to coagulating any albumen which may exist in the urine, cause the evolution of carbonic acid in large quantities. The author has never succeeded in finding sugar. The colour of the urine has been supposed by the laity, and even by members of the profession, to be due to an intermixture of blood; indeed many writers assert that they have actually found blood-corpuscles under the microscope. Among these are Mr. Hogoulin, at Bourbon, M. Bories and M. Pellarin, all of whom the author quotes. The last-named gentleman says he never failed to find blood-disks, except when the urine was alkaline. Unfortunately American observers seem to have had recourse to the microscope in very few instances, but to have contented themselves with a simple inspection of the urine. Prof. N. F. Lupton, of the Southern University, states that it was found in two specimens submitted to him for examination, and hence Drs. T. C. Osborn and Michel, who quote him, conclude that it exists in every case.[1]

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[1] The following is Prof. Lupton's report of his examination of the urine: "In quantity the secretion rarely varied from a healthy standard, but all its other features were continually changing. At one time it would be a true hæmaturia with albuminous deposit, at another amber-coloured and full of dumb-bell crystals, beautifully distinct, and again limpid, with a splendid iridescent pellicle

M. Berenger-Feraud repeated M. Pellarin's experiments, using all the precautions which he recommends, in order to insure success, and although he was aided in his investigations by M. Trouette, *Pharmacien de la première classe*, and an accomplished chemist, utterly failed in finding anything else but epithelial cells and casts of the uriniferous tubules. MM. Hogoulin, Bories, and Pellarin are convinced that the colour of the urine is not due to the presence of bile, because no colour-tests are obtained when nitric or hydrochloric acid is added to it. The author believes, on the other hand, that the usual reactions with these acids are prevented by the albumen which the urine contains, and which must first be got rid of before bile can be detected. Inasmuch as his theory rests upon the presence of biliary colouring matters in the urine, we intend to give his method of proceeding in his own words. He chooses for his purpose urine freshly passed. It was very dark red, almost black in colour, resembling very dark Malaga wine.

"This urine," he says, "the specific gravity of which was 1020 at the time of emission, let fall almost immediately a grayish, flaky deposit, which became by degrees considerable in amount. The precipitate and the supernatant liquid examined repeatedly with the microscope never revealed the presence of blood-corpuscles. A portion of this urine treated with an equal volume of alcohol at 90°, gave an abundant precipitate. This was filtered and evaporated at a gentle heat until it was reduced to a very small bulk, when a blackish-brown substance was precipitated. This precipitate was collected on a filter, washed with water, and then treated with chloroform, which yielded, 1, a yellow liquid; 2, an insoluble substance. The chloroformic solution of yellow colour was then agitated with a solution of caustic soda, which deprived the chloroform of its yellow colour. An equal volume of alcohol at 90° was then added to the alkaline solution, which was then treated with concentrated nitroso-nitric acid, the effect of which was to produce at first a green colour, then blue, violet, dark red, and finally a dirty yellow. Another portion of the chloroformic solution, evaporated to dryness in a watchglass, yielded an amorphous orange-red substance, without a trace of crystallization, even when examined under the microscope; we therefore think ourselves justified," he says, "in asserting that our experiments have shown the presence of bilirubine.

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floating upon the surface. When it was hæmaturic, *blood disks* and tube casts were more or less abundant in the field of the microscope, and albumen and clots would fall to the bottom of the vessel. I mention these particulars because several intelligent physicians have expressed doubts as to the bloody character of the secretion. Only once did the urine fail to redden litmus paper, and that was during the presence of the triple phosphates; it was then slightly alkaline. The specific gravity varied in the different specimens from 1010 to 1030, being highest in the hæmaturic, and lowest in the phosphatic. Sometimes the changes would follow each other in rapid succession during the day."

Since this review was in print Dr. J. M. Da Costa, Professor of Practice of Medicine in the Jefferson Medical College, has informed the writer that he has examined microscopically three specimens of urine presenting the characters described in the text, and in all of them detected abundance of blood-corpuscles. In two cases the specimens were sent to him from a distance, but were in a good state of preservation when examined; in the other case the patient, who had in other respects a typical attack of malarial fever, was under his own care in this city, although the disease had been contracted in Maryland. Nothing is more likely than that hemorrhage may occur from the kidneys, as a consequence of the congestion resulting from the increased functional activity of these organs in separating the bile from the blood. Indeed M. Berenger-Feraud admits this, contending, however, that, since blood was not detected in the numerous examinations he made, we must look further for the cause of the discolouration of the urine. In fact, Dr. Lupton's expression that the blood disks were more or less abundant does not convey to our mind the impression that they were present in such quantities as to explain this most striking feature of the disease.

"The precipitate, which was insoluble in chloroform, dissolved readily in alcohol. It was soluble also in a solution of caustic soda and in aqua ammoniæ. The soda solution yielded with muriatic acid a yellowish-brown precipitate. The ammoniacal solution yielded, when treated with calcium chloride, a rusty-coloured precipitate."

These experiments prove, the author believes, the presence of bilifuscine.

"The presence of the biliary acids was recognized in the following manner: A certain quantity of urine was evaporated to a syrupy consistence, then treated with alcohol at 90°, and afterwards filtered. The portion passing through the filter was then again evaporated and treated with absolute alcohol. Again filtered, and the alcohol eliminated by means of heat, it was treated with a little water. Acetate of lead being added to this liquid, a precipitate was obtained, which, after being washed in a solution of sodium carbonate, was boiled for a long time. The liquid thus obtained was evaporated to dryness, and the residue treated with alcohol, which was afterwards evaporated. The substance left after evaporation was dissolved in water and submitted to Pettenkofer's test, when a magnificent violet colour was produced."

After having thus shown that the colour of the melanuric urine is due to the presence of biliary principles, M. Berenger-Feraud says that to make assurance doubly sure, he has added bile, taken from the body of an individual who died of this disease, to normal urine, with the effect of producing a liquid indistinguishable from melanuric urine. He adds, also, that, if the blood be treated with the same reagents, it will also be found to contain bile.

From these experiments the authors think the following conclusions established:—

1. The urine in melanuric bilious fever does not contain a trace of blood, and its very remarkable colour is due to the presence of a large quantity of biliary matters.[1]
2. The colouring matters which it contains, and which give it a dark tint resembling Malaga wine or the infusion of coffee, are bilirubine, and bilifuscine, to which must be added the biliary acids.
3. Bile is also found in the blood as it leaves the liver.

The stools and vomited matters were also carefully examined and found to contain a notable quantity of bile.

The only diseases likely to be confounded with the one under notice are acute atrophy of the liver and yellow fever. In the former, which is a rare disease, there is an entire absence of fever; the pulse is slow rather than frequent. It has not, moreover, an intermittent or remittent character, and does not yield to quinia. Nitric and muriatic acids show the presence of bile in the urine, and there is diminution of the hepatic dulness. It is scarcely necessary to add that the post-mortem appearances in the two diseases are very different. We will not give the means by which we are enabled to make a differential diagnosis between melanuric bilious fever and yellow fever, although the author does this very fully and carefully. In the course of this review we have so frequently contrasted the symptoms of these diseases, that it would be superfluous to do it now.

Symptoms of evil omen are, a violent chill or syncope at the commencement, and nervous symptoms of all kinds, such as jactitation and wild delirium. A very sudden, irregular, or delayed appearance of the jaundice is likewise unfavourable, as are also suppression of urine or great diminution in quantity, a persistence of the dark colour, constant vomiting, which continues in spite of remedies, intense lumbar pains, and hiccough. It must not be forgotten that occasionally fatal cases begin insidiously.

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[1] The author adds, that upon his return to Europe, he submitted several specimens of melanuric urine to M. Bouchardat. This learned professor, whose attainments justly give weight to his opinions on scientific matters, and who possesses moreover unusual skill in the analysis of the urine, said, after the examination of the liquid, that the discolouration was due to the presence of bile, and that in a number of similar specimens which he had analyzed he had never found anything else but bile.

We arrive now at one of the most important subjects in the book, the treatment of the disease. From what has preceded, our readers must of course be aware that M. Berenger-Feraud regards the paroxysm of melanuric bilious fever as only differing from that of other forms of pernicious fever in the excessive secretion of bile, replacing the more usual sweating, or the choleraic discharges. Holding this view of the nature of the disease, it is not surprising that he should regard quinia as the sheet anchor in its treatment, and that he should look with disfavour upon all plans of treatment which have for their object the promotion of the secretion of bile. The symptoms are largely due to the absorption of bile, and this is very likely to be increased by anything which augments the quantity of bile in the intestines. Moreover, experience has shown him that while the jaundice is increased when mercury is administered, it is diminished under the use of quinia.

We intend now to present to our readers, in as few words as possible, the plan of treatment which this author recommends, and which long experience has shown him to be most useful. We shall afterwards review the methods which have been proposed by others, and especially by physicians in our Southern States.

Called to a case of the disease, in the forming stage, M. Berenger-Feraud, with the view of correcting the saburral condition of the stomach, administers either an evacuant, under which head are included emetics and cathartics, or an opiate. An emetic should only be used in first or very mild attacks, and if the patient have been in good health before it. Ipecac. is the best of all emetics; its action should be promoted by copious draughts of warm water. Its effect is, after it has itself ceased to act, to arrest the vomiting; once used, it is better not to recur to it. Since purgation is less distressing and debilitating than emesis, a cathartic is recommended in relapses and for feeble subjects. One not likely to nauseate should be chosen. He was first induced to try opiates in this condition by reading the memoir of Husson in the *Annuaire Médico-Chirurgical des Hôpitaux et Hospices de Paris*, in which reference is made to the use of this drug by Hernandez in large doses—four grammes of the wine of opium daily—at the beginning of bilious fever, for the purpose of arresting the vomiting. The dose recommended is large, and inasmuch as death in one case was ascribed to it by Husson, the author prescribes the *sirop* de diacorde—a preparation not materially differing from paregoric, to be taken as follows: Thirty or forty grammes are to be mixed with a hundred grammes of water. Of this a tablespoonful is to be given every hour. When thus administered sleep will be induced before the patient will have taken a poisonous dose of the drug. He has used this prescription more than three hundred times in various conditions, in which there was hepatic and gastric disorder, and has always found it beneficial; in fact he is convinced that it is quite as efficacious as an emetic, and vastly more agreeable to the patient. In the fever under consideration, his success has not been quite so remarkable, but still sufficiently great to justify him in recommending the treatment most cordially to the notice of the profession. He seems to use the opiate or an emetic indifferently, and indeed sometimes gives them in the same case. He says, however, that his experience leads him to advise the use of the former as the rule, and that of the latter as the exception, simply because, in hot climates, it is necessary to avoid debilitating remedies. A still further objection to the emetic is, that it is likely to increase the already existing irritation of the stomach.

As soon as the vomiting has been checked and the fever moderated, or, in other words, as soon as possible, the patient should be placed under the influence of quinia. This should always be given in solution, and to insure its retention by the stomach, a few drops of laudanum should be added to it. If the medicine is rejected, it should be immediately repeated, and if again rejected, it should be given by the rectum, which, according to Briquet, absorbs it more readily than does the stomach. The author also speaks well of the hypodermic administration of quinia, although he has never so employed it. He condemns its application to blistered surfaces as useless. The dose, when given by the mouth, should be from two to two and a half grammes; this is to be followed in from three to twelve hours by a gramme in two doses. A gramme or more, in proportion to the severity of the case, is to be given next day, and to be continued daily until the disease is cured.

Certain symptoms are so prominent that it will be necessary to address remedies especially to them. Such a one is vomiting. When opium or an emetic fails to arrest this, recourse may be had in turn to cherry-laurel water, essence of peppermint, effervescing lemonade, champagne, ice, and hot drinks, although to none of these does the author attribute any positive curative power. In some cases, in which every other means fail, he has seen good result from the administration of teaspoonfuls of water given every minute. In others it will be necessary to give the stomach entire rest, and to give food, which must consist of milk and nourishing broths, as well as medicine, by the rectum. The pains in the loins and in the region of the liver are often distressing, and are best relieved by emolient cataplasms and by embrocations with sweet oil. Blisters have been used for this purpose, but when applied to the back vastly increased the sufferings of the patient. The adynamic condition which succeeds the fever requires the continuance of the quinia, which may be given in smaller quantities, care being taken to forestall the tendency to relapse by giving it in full doses on every seventh day. But it should never be replaced by the wine of bark, which is a preparation of uncertain strength, and which contains so much alcohol as to be objectionable on that account; many of the soldiers regarding it as a more agreeable "bitter" than can be purchased at the suttler's.

In his hands, and in those of M. Bourgarel, the plan of treatment just detailed has been eminently successful, M. Bourgarel having had eighteen successive cases all ending in recovery. The same treatment has been adopted in this country and notably by Dr. Norcom, and with very similar results. "I have treated," this gentleman says, "eleven cases of this disease, ten of which recovered. My first case, the one that died, sank from pure exhaustion within an hour after I first saw her." He gives somewhat larger doses of quinia, and substitutes for the opium by the mouth, from $\frac{1}{4}$ to $\frac{1}{3}$ of a grain of acetate of morphia administered hypodermically, claiming for it the power of checking the vomiting in a wonderfully short space of time. No other plan of treatment has been attended by anything like the same amount of success, and it appears to us the most rational that has been proposed, because it is directed against the cause of the disease, and not against an effect of it. But it is by no means generally followed, our southern brethren, with very few exceptions, regarding it as necessary to institute some preliminary treatment before bringing the patient under the influence of quinia. Calomel, therefore, still continues to be given, and with two objects in view—1st, as a purgative; 2d, as an alterative. To fulfil the first indication it has been given in very large doses, from 20 to 30 grains As an alterative it has been prescribed in divided doses, in some cases continued until salivation is produced; in others it is not pushed so far. M. Berenger-Feraud objects to either use of calomel. As a purgative its action may be excessive, and, therefore, debilitating to the patient. In smaller doses it is, he thinks, an irritant to the liver, which ought to prevent its use by prudent physicians in tropical countries. The stomatitis produced by it is, moreover, very inconvenient, interfering very decidedly with the nutrition of the patient, and demanding very often a large share of the physician's attention. The author's objections to the use of mercury are, however, based on better grounds than mere theoretical objections. In looking over the records of the hospital at Gorée, he found that next to compound fractures the condition that caused the retention of patients the longest was syphilis, and that after this came hepatitis. He found, moreover, that a large proportion of patients suffering from hepatitis had previously been treated for syphilis, and that under these circumstances the disease was very apt to terminate in suppuration. He has also observed that the former disease is very apt to follow any other disease that has been treated with mercury, he therefore avoids this remedy if the patient must remain in the colony after his convalescence. The author's researches have likewise convinced him that patients treated with it are just as likely as any others, and perhaps more than others, to suffer from relapse, a fact which proves that it has no real influence over the disease.

These objections are not altogether of recent origin. When speaking of the treatment of fevers, Twining[1] tells us that the use of mercury must be abandoned if the enlargement of the spleen supervenes, and in another part of his book says that the benefit of salivation is problematical. Morehead[2] is even more decided in his condemnation of the remedy. "Full doses of calomel," he says, "and the free use of purgatives form no part of the treatment of remittent fever with jaundice. On the contrary, they are likely to cause aggravation of the symptoms and to hurry on the period of prostration." He refers to the experiments of Dr. Murray upon dogs as proving that calomel exercises an irritant action on the gastro-intestinal mucous membrane, and asserts that many of the effects of calomel have been described as symptoms of the disease. "For these reasons," he continues, "I am of opinion that an endeavour to induce mercurial influence in remittent fever is erroneous in theory and of no value in practice." We might multiply quotations still further, but shall content ourselves with mentioning, as authors holding very similar views to those of Twining and Morehead, the following: Copeland,[3] Robert Jackson,[4] Geddes[5], and in more recent times Maclean.[6] Much difference of opinion exists among southern physicians as to the propriety of giving opium in this disease. Thus, while we find Dr. Starley agreeing with Dr. Norcom that it is a useful remedy, Drs. Michel and T. C. Osborn condemn it as likely to do harm. Astringents have of course been recommended by those who believe that the alteration of the urine depends upon hæmaturia, but they are, it is needless to say, not indicated if it is due to an admixture of bile. Diuretics are recommended by Dr. J. D. Osborn to relieve the supposed congestion of the kidneys.

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[1] Disease of Bengal. Calcutta, 1835, pp. 452 and 479.

[2] Clinical Researches on Diseases in India. London, 1856, vol. i. p. 197.

[3] Medical Dictionary, vol. i. p. 928.

[4] Sketch of the History and Cure of Febrile Diseases, 1817, p. 243.

[5] Clinical Illustrations of the Diseases of India.

[6] Reynolds's System of Medicine, vol. i. p. 80.

We have little to add in the way of criticism of M. Berenger-Feraud's views of the pathology of this disease. These rest upon observations and experiments which we feel sure were carefully made, and must therefore, it seems to us, be accepted, until they are shown by further observation to be incorrect. It is easier, indeed, to understand that hemorrhage should take place from the kidneys during the paroxysms than that the bile which is secreted in excess at this time should be immediately absorbed and excreted with the urine. But no theory can stand if opposed to facts, and we must therefore temporarily at least abandon that which assumes the existence of hæmaturia in this disease. It gives us pleasure to add, that at least one American physician suggested long before the publication of this work that the discolouration of the urine might be due to the presence of bile. "It appears to me, however," Prof. Joynes writes, "very improbable that the red colour of the urine was due to admixture of blood, as there is no mention of hemorrhage from any other organ than the kidney. That the mere presence of the colouring principle of the bile may render the urine red is in fact well known."

M. Berenger-Feraud deserves the thanks of the profession. Working in a country where the climate renders all exercise of the intellect difficult, and cut off from all books of reference, he has nevertheless produced a book which is one of the best of the kind we are acquainted with.

J. H. H.

ART XXI.—*The Physiology of Man; designed to represent the existing State of Physiological Science, as applied to the Functions of the Human Body.* By AUSTIN FLINT, Jr., M.D., Professor of Physiology and Physiological Anatomy in the Bellevue Hospital Medical College, New York; Physician to the Bellevue Hospital, etc. etc. etc. In five volumes. Vol. V. (with a general index to the five volumes). Special Senses; Generation. 8vo. pp. 517. New York: D. Appleton & Co., 1874.

THIS is the fifth and concluding volume of the physiological work upon which Prof. Austin Flint, Jr., has been engaged during the past eleven years. The first volume appeared in 1866, the second in 1867, the third in 1870, and the fourth in 1872. As they were issued, we presented from time to time, in the pages of this Journal, a brief analysis of their contents. We propose to perform the same task for the present volume, in which, in a compass of 463 pages, are discussed the special senses and the function of reproduction. The remaining 54 pages are wholly taken up with two indices, a special one for the volume itself, and a general one for the entire work.

Chapter I. is devoted to a general consideration of the special senses, and a particular account of the sense of touch. Though, in view of the well-known researches of Dr. Brown-Séquard, Dr. Flint recognizes the possibility of impressions of touch, titillation, temperature, and pain